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(FILE 'HOME' ENTERED AT 12:01:36 ON 28 JAN 2002)

FILE 'BIOSIS' ENTERED AT 12:01:48 ON 28 JAN 2002

L1 56349 S DROSOPHILA
L2 412 S L1 AND (HYBRID DYSGENESIS)
L3 0 S L2 AND (GENE IDENTIFICATION)
L4 136 S L2 AND (P ELEMENT)
L5 0 S L4 AND (MODULATING EXPRESSION)
L6 133 S L4 AND PD<=19990812

d l4 ti abs ibib 27 33 35 39 55 57 86 109 114 115 135 136

L4 ANSWER 27 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI **Drosophila P element**: Transposition, regulation and evolution.
ACCESSION NUMBER: 1995:63332 BIOSIS
DOCUMENT NUMBER: PREV199598077632
TITLE: **Drosophila P element**: Transposition, regulation and evolution.
AUTHOR(S): Coen, Dario (1); Lemaitre, Bruno; Delattre, Marion (1); Quesneville, Hadi (1); Ronsseray, Stephane (1); Simonelig, Martine (1); Higuet, Dominique (1); Lehmann, Monique (1); Montchamp, Catherine (1); Nouaud, Danielle (1); Anxolabehere, Dominique (1)
CORPORATE SOURCE: (1) Dep. Dynamique du Genome et Evol., Inst. Jacques Monod, Tour 43, 2 place Jussieu, F-75251 Paris Cedex 05 France
SOURCE: Genetica (Dordrecht), (1994) Vol. 93, No. 1-3, pp. 61-78. ISSN: 0016-6707.
DOCUMENT TYPE: General Review
LANGUAGE: English

L4 ANSWER 33 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI P transposable element in **Drosophila** melanogaster: An horizontal transfer.
AB The P transposable element family in **Drosophila** melanogaster is responsible for the syndrome of **hybrid dysgenesis** which includes chromosomal rearrangements, male recombination, high mutability and temperature sensitive agametic sterility (called gonadal dysgenesis sterility). **P element** activity is controlled by a complex regulation system, encoded by the elements themselves, which keeps their transposition rate low within the strain bearing **P elements** and limits copy number by genome. A second regulatory mechanism, which acts on the level of RNA processing, prevents P mobility to somatic cells. The oldest available strains, representing most major geographical regions of the world, exhibited no detectable hybridization to the **P-element**. In contrast, all recently collected natural populations that were tested carried **P-element** sequences. The available evidence is consistent with the hypothesis of a worldwide **P-element** invasion of D. melanogaster during the past 30 years. Timing and direction of the invasion are discussed. The lack of **P-element** in older strains of **Drosophila** melanogaster as well as in the species must closely related to **Drosophila** melanogaster, suggests that P entered the **Drosophila** melanogaster genome recently, probably by horizontal transfer from an other species. The analysis of **P-element** elsewhere in the genus **Drosophila** reveals that several more distantly related species carried transposable elements with sequences quite similar to P. The species with the best-matching **P-elements** is D. willistoni. A **P-element** from this species was found to match all but one of the 2907 nucleotides of the **Drosophila** melanogaster **P-element**. The phylogenic distributions and the likely horizontal transferts of the two other **Drosophila** transposable elements are discussed.
ACCESSION NUMBER: 1993:319678 BIOSIS
DOCUMENT NUMBER: PREV199396028028
TITLE: P transposable element in **Drosophila** melanogaster: An horizontal transfer.
AUTHOR(S): Anxolabehere, Dominique
CORPORATE SOURCE: Institut Jacques-Monod, Laboratoire de Dynamique du Genome et Evolution, Tour 42, 2 Place Jussieu, 75251 Paris Cedex 05 France

SOURCE: Comptes Rendus des Seances de la Societe de Biologie et de ses Filiales, Vol. 186, No. 6, pp. 641-655.
ISSN: 0037-9026.
DOCUMENT TYPE: Article
LANGUAGE: French
SUMMARY LANGUAGE: French; English

L4 ANSWER 35 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Structure and expression of **hybrid dysgenesis**-induced alleles of the ovarian tumor (otu) gene in **Drosophila** melanogaster.
AB Mutations at the ovarian tumor (otu) gene of **Drosophila** melanogaster cause female sterility and generate a range of ovarian phenotypes. Quiescent (QUI) mutants exhibit reduced germ cell proliferation; in oncogenic (ONC) mutants germ cells undergo uncontrolled proliferation generating excessive numbers of undifferentiated cells; the egg chambers of differentiated (DIF) mutants differentiate to variable degrees but fail to complete oogenesis. We have examined mutations caused by insertion and deletion of **P elements** at the otu gene. The **P element** insertion sites are upstream of the major otu transcription start sites. In deletion derivatives, the **P element**, regulatory regions and/or protein coding sequences have been removed. In both insertion and deletion mutants, the level of otu expression correlates directly with the severity of the phenotype: the absence of otu function produces the most severe QUI phenotype while the ONC mutants express lower levels of otu than those which are DIF. The results of this study demonstrate that the diverse mutant phenotypes of otu are the consequence of different levels of otu function.
ACCESSION NUMBER: 1993:186007 BIOSIS
DOCUMENT NUMBER: PREV199395096457
TITLE: Structure and expression of **hybrid dysgenesis**-induced alleles of the ovarian tumor (otu) gene in **Drosophila** melanogaster.
AUTHOR(S): Sass, Georgette L. (1); Mohler, J. Dawson; Walsh, Rosemary C. (1); Kalfayan, Laura J. (1); Searles, Lillie L.
CORPORATE SOURCE: (1) Dep. Biochem. Biophys., The Univ. N.C., Chapel Hill, NC 27599-3280 USA
SOURCE: Genetics, (1993) Vol. 133, No. 2, pp. 253-263.
ISSN: 0016-6731.
DOCUMENT TYPE: Article
LANGUAGE: English

L4 ANSWER 39 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI VISIBLE MUTATIONS INDUCED BY P-M **HYBRID DYSGENESIS** IN **DROSOPHILA**-MELANOGASTER RESULT PREDOMINANTLY FROM **P ELEMENT** INSERTIONS.
AB This study supplied no evidence that P-M **hybrid dysgenesis** is a general release mechanisms for transposon movement. Newly induced mutations (23 singed, three yellow, and one white) were generated by **hybrid dysgenesis** and were molecularly analyzed for the presence or absence of **P element** insertions. Only one dysgenically-induced insertion mutation out of 27 analyzed was the result of a non-P insert; this frequency is not statistically above the non-dysgenic control level. Thus, it appears that individual transposable elements families are independently regulated.
ACCESSION NUMBER: 1992:501427 BIOSIS
DOCUMENT NUMBER: BA94:119952
TITLE: VISIBLE MUTATIONS INDUCED BY P-M **HYBRID DYSGENESIS** IN **DROSOPHILA**-MELANOGASTER RESULT PREDOMINANTLY FROM **P ELEMENT**

INSERTIONS.
AUTHOR(S): NORRIS E S; WOODRUFF R C
CORPORATE SOURCE: DEP. BIOL., UNIV. MICHIGAN, ANN ARBOR, MI 48109, USA.
SOURCE: MUTAT RES, (1992) 269 (1), 63-72.
CODEN: MUREAV. ISSN: 0027-5107.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 55 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI DYNAMICS OF P-M **HYBRID DYSGENESIS** IN P-TRANSFORMED LINES OF **DROSOPHILA**-SIMULANS.
AB An autonomous **P element** from **Drosophila** melanogaster was introduced by microinjection into the germ line of its sibling species, D. simulans. The invasion kinetics of **P elements** was studied in seven independent lines over 60 generations, using gel blotting, in situ hybridization, and dysgenic crosses. Some of the main phenotypic and molecular characteristics of P-M **hybrid dysgenesis** were observed, i.e., gonadal dysgenesis (GD sterility), chromosome rearrangements, and the occurrence of degenerate **P elements**. At least four lines reached a steady state with complete or nearly complete **P-element** regulation but with a moderate number of **P elements** (10-24 per haploid genome) and P activity (10-35% GD sterility). This failure to obtain strong P strains in D. simulans could be due to experimental conditions, a host-dependent component of P transposition, or more severe selection against the deleterious effects of this transposon.
ACCESSION NUMBER: 1990:239052 BIOSIS
DOCUMENT NUMBER: BA89:126005
TITLE: DYNAMICS OF P-M **HYBRID DYSGENESIS** IN P-TRANSFORMED LINES OF **DROSOPHILA**-SIMULANS.
AUTHOR(S): MONTCHAMP-MOREAU C
CORPORATE SOURCE: LAB. GENET. POPULATIONS, UA CNRS 693, UNIV. P. M. CURIE, 4 PLACE JUSSIEU, 75005 PARIS, FR.
SOURCE: EVOLUTION, (1990) 44 (1), 194-203.
CODEN: EVOLAO. ISSN: 0014-3820.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 57 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI REPRESSION OF **P ELEMENT**-MEDIATED **HYBRID DYSGENESIS** IN **DROSOPHILA**-MELANOGASTER.
AB Inbred lines derived from a strain called Sexi were analyzed for their abilities to repress **P element**-mediated gonadal dysgenesis. One line had high repression ability, four had intermediate ability and two had very low ability. The four intermediate lines also exhibited considerable within-line variation for this trait; furthermore, in at least two cases, this variation could not be attributed to recurring **P element** movement. Repression of gonadal dysgenesis in the hybrid offspring of all seven lines was due primarily to a maternal effect; there was no evidence for repression arising de novo in the hybrids themselves. In one of the lines, repression ability was inherited maternally, indicating the involvement of cytoplasmic factors. In three other lines, repression ability appeared to be determined by partially dominant or additive chromosomal factors; however, there was also evidence for a maternal effect that reduced the expression of these factors in at least two of the lines. In another line, repression ability seemed to be due to recessive chromosomal factors. All seven lines possessed numerous copies of a particular **P element**, called KP, which has been hypothesized to produce a polypeptide repressor of gondal dysgenesis. This hypothesis, however, does not explain why the inbred Sexi lines varied so much in their repression abilities. It is suggested that some of

this variation may be due to differences in the chromosomal position of the KP elements, or that other nonautonomous **P elements** are involved in the repression of **hybrid dysgenesis** in these lines.

ACCESSION NUMBER: 1990:197468 BIOSIS
DOCUMENT NUMBER: BA89:104139
TITLE: REPRESSION OF **P ELEMENT**-MEDIATED **HYBRID DYSGENESIS** IN **DROSOPHILA** -MELANOGASTER.
AUTHOR(S): SIMMONS M J; RAYMOND J D; RASMUSSON K E; MILLER L M; MCLARNON C F; ZUNT J R
CORPORATE SOURCE: DEP. GENETICS CELL BIOLOGY, UNIV. MINNESOTA, ST. PAUL, MINNESOTA 55108-1095.
SOURCE: GENETICS, (1990) 124 (3), 663-676.
CODEN: GENTAE. ISSN: 0016-6731.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 86 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI MOLECULAR ANALYSIS OF THE NEUROGENIC LOCUS MASTERMIND OF **DROSOPHILA**-MELANOGASTER.
AB The neurogenic loci comprise a small group of genes which are required for proper division between the neural and epidermal pathways of differentiation within the neuroectoderm. Loss of neurogenic gene function results in the misrouting of prospective epidermal cells into neuroblasts. A molecular analysis of the neurogenic locus mastermind (mam) has been initiated through transposon tagging with **P elements**. Employing the Harwich strain as the source of P in a **hybrid dysgenesis** screen, 6000 chromosomes were tested for the production of lethal mam alleles and eight mutations were isolated. The mam region is the site of residence of a **P element** in Harwich which forms the focus of a chromosome breakage hotspot. Hybrid dysgenic induced mam alleles elicit cuticular and neural abnormalities typical of the neurogenic phenotype, and in five of the eight cases the mutants appear to retain a **P element** in the cytogenetic region (50CD) of mam. Utilizing **P element** sequence as probe, mam region genomic DNA was cloned and used to initiate a chromosome walk extending over 120 kb. The physical breakpoints associated with the hybrid dysgenic alleles fall within a 60-kb genomic segment, predicting this as the minimal size of the mam locus barring position effects. The locus contains a high density of repeated elements of two classes; opa (CAX)n and (dC-dA)n. (dG-dT)n. A preliminary study of the transcriptional activity of the mam region is presented.

ACCESSION NUMBER: 1988:243080 BIOSIS
DOCUMENT NUMBER: BA85:121482
TITLE: MOLECULAR ANALYSIS OF THE NEUROGENIC LOCUS MASTERMIND OF **DROSOPHILA**-MELANOGASTER.
AUTHOR(S): YEDVOBNICK B; SMOLLER D; YOUNG P; MILLS D
CORPORATE SOURCE: DEP. BIOL., EMORY UNIV., ATLANTA, GEORGIA 30322.
SOURCE: GENETICS, (1988) 118 (3), 483-498.
CODEN: GENTAE. ISSN: 0016-6731.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 109 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI MOLECULAR CLONING OF SUPPRESSOR OF SABLE A **DROSOPHILA** -MELANOGASTER TRANSPOSON-MEDIATED SUPPRESSOR.
AB A **hybrid dysgenesis**-induced allele [su(s)W20] associated with a **P-element** insertion was used to clone sequences from the su(s) region of **Drosophila** melangaster by means of the transposon-tagging technique. Cloned sequences were used to probe restriction enzyme-digested DNAs from 22 other su(s) mutations.

None of three X-ray-induced or six ethyl methanesulfonate-induced su(s) mutations possess detectable variations. Seven spontaneous, four **hybrid dysgenesis**-induced, and two DNA transformation-induced mutations were associated with insertions within 2.0 kilobases (kb) of the su(s)W20 **P-element** insertion site. When the region of DNA that included the mutational insertions was used to probe poly(A)+ RNAs, a 5-kg message was detected in wild-type RNA that was present in greatly reduced amounts in two su(s) mutations. By using strand-specific probes, the direction of transcription of the 5-kb message was determined. The mutational insertions lie in DNA sequences near the 5' end of the 5-kb message. Three of the seven spontaneous su(s) mutations are associated with gypsy insertions, but they are not suppressible by su(Hw).

ACCESSION NUMBER: 1986:279496 BIOSIS
DOCUMENT NUMBER: BA82:23359
TITLE: MOLECULAR CLONING OF SUPPRESSOR OF SABLE A **DROSOPHILA**-MELANOGASTER TRANSPOSON-MEDIATED SUPPRESSOR.
AUTHOR(S): CHANG D-Y; WISELY B; HUANG S-M; VOELKER R A
CORPORATE SOURCE: LAB. GENETICS, NATIONAL INST. ENVIRONMENTAL HEALTH SCI., RESEARCH TRIANGLE PARK, NORTH CAROLINA 27709.
SOURCE: MOL CELL BIOL, (1986) 6 (5), 1520-1528.
CODEN: MCEBD4. ISSN: 0270-7306.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 114 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI **P-ELEMENT**-INDUCED CONTROL MUTATIONS AT THE R GENE OF **DROSOPHILA**-MELANOGASTER.
AB The P-M **hybrid dysgenesis** system was used to produce five putative regulatory mutations at the rudimentary locus, r. All five mutations were the result of insertions at the 5' end of the gene, upstream of the proposed start of transcription. All of the mutants displayed a leaky wing phenotype, and four of the mutants showed an uncoupling of the wing and female-sterility phenotypes, suggesting that they altered the normal spatial and temporal expression of the r gene. Four of the insertions were **P elements**. The fifth insertion, which was larger than an intact **P element**, consisted of a small **P element** connected to non-**P-element** DNA. Two of the mutants produced very little r transcript in adult females and were clustered 80 to 150 base pairs upstream of the start of transcription. The other three mutants had higher levels of r transcript in adult females and were clustered 440 to 500 base pairs upstream of the start of transcription. All of the data suggest that the insertions are in a 5' noncoding region of the r gene involved in the control of its spatial and temporal expression.

ACCESSION NUMBER: 1986:103093 BIOSIS
DOCUMENT NUMBER: BA81:13509
TITLE: **P-ELEMENT**-INDUCED CONTROL MUTATIONS AT THE R GENE OF **DROSOPHILA**-MELANOGASTER.
AUTHOR(S): TSUBOTA S; ASHBURNER M; SCHEDL P
CORPORATE SOURCE: DEP. BIOL., PRINCETON UNIV., PRINCETON, N.J. 08544.
SOURCE: MOL CELL BIOL, (1985) 5 (10), 2567-2574.
CODEN: MCEBD4. ISSN: 0270-7306.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 115 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI **HYBRID DYSGENESIS** IN **DROSOPHILA**-MELANOGASTER NATURE AND INHERITANCE OF **P ELEMENT** REGULATION.
AB The genetic determination of the control of resistance or susceptibility to germ line changes mediated by **P elements** was

studied in two strains and in derivatives of crosses between them. One strain, characterized as true M, completely lacked **P elements**. The second strain, pseudo-M (M'), carried a number of **P elements**, but these did not have the potential to induce the gonadal sterility that is associated with P-M **hybrid dysgenesis**. Individuals from the true M strain were invariably unable to suppress P factor activity (i.e., all daughters of outcrosses of M females and P males were sterile). In contrast, individuals from the M' strain showed variable degrees of suppression that were manifested in a wide range of gonadal sterility frequencies in standard tests. This continuous distribution pattern was reproducible for more than 25 generations. -The results of the genetic analysis indicate that a strain with a variable degree of suppression of gonadal dysgenesis is not necessarily in a transient state between the extreme conditions of P and M cytotype. A large variance in the ability to suppress gonadal dysgenesis with a mean value intermediate between the extremes of P and M cytotype may be a relatively stable strain characteristic. No reciprocal cross effect was observed in the suppression of sterility of F1 females from M .times. M' matings. Thus, the existence of M' strains indicates of Mendelian component in P **element regulation** and suggests that cytotype, which has an extrachromosomal aspect, may be only one of perhaps several mechanisms involved in regulation. Analysis of the effects of individual chromosomes from the M' strain showed that each chromosome contributed to the reduction of gonadal dysgenesis in the progeny of test matings. The results are consistent with a one-component titration model for P **element regulation**.

ACCESSION NUMBER: 1986:103030 BIOSIS
DOCUMENT NUMBER: BA81:13446
TITLE: **HYBRID DYSGENESIS** IN **DROSOPHILA** -MELANOGASTER NATURE AND INHERITANCE OF **P ELEMENT** REGULATION.
AUTHOR(S): KIDWELL M G
CORPORATE SOURCE: DEP. ECOL. EVOLUTIONARY BIOL., UNIV. ARIZ., TUCSON, ARIZ. 85721.
SOURCE: GENETICS, (1985) 111 (2), 337-350. CODEN: GENTAE. ISSN: 0016-6731.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 135 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI THE MOLECULAR BASIS OF P-M **HYBRID DYSGENESIS** THE NATURE OF INDUCED MUTATIONS.
AB The molecular nature of mutations arising in dysgenic hybrids between P and M **Drosophila** melanogaster strains was investigated. Seven independent mutations at the white locus were examined, and these fell into 2 classes on the basis of their genetic and structural properties. The 5 mutations comprising the 1st class were caused by DNA insertions of 0.5, 0.5, 0.6, 1.2 and 1.4 kb [kilobase], respectively. The DNA insertions in 4 of these mutations were examined in detail. Although heterogeneous in size and pattern of restriction enzyme sites, they were homologous in sequence. Members of this sequence family are referred to as **P elements**. Mutations caused by **P elements** appeared to be stable in the P cytotype, but had reversion rates greater than 10-3 in the M cytotype. Phenotypic reversion to wild-type was accompanied by excision of the **P element**. The 2 mutations comprising the 2nd class were caused by insertion of the 5.0 kb copia element and appeared to be stable in both P and M cytotypes.

ACCESSION NUMBER: 1983:191533 BIOSIS
DOCUMENT NUMBER: BA75:41533
TITLE: THE MOLECULAR BASIS OF P-M **HYBRID DYSGENESIS** THE NATURE OF INDUCED MUTATIONS.
AUTHOR(S): RUBIN G M; KIDWELL M G; BINGHAM P M

CORPORATE SOURCE: DEP. EMBRYOL., CARNEGIE INST. WASHINGTON, 115 W. UNIVERSITY PARKWAY, BALTIMORE, MD. 21210.
SOURCE: CELL, (1982) 29 (3), 987-994.
CODEN: CELLB5. ISSN: 0092-8674.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L4 ANSWER 136 OF 136 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI THE MOLECULAR BASIS OF P-M **HYBRID DYSGENESIS** THE ROLE OF THE **P ELEMENT** A P STRAIN SPECIFIC TRANSPOSON FAMILY.
ACCESSION NUMBER: 1983:33593 BIOSIS
DOCUMENT NUMBER: BR24:33593
TITLE: THE MOLECULAR BASIS OF P-M **HYBRID DYSGENESIS** THE ROLE OF THE **P ELEMENT** A P STRAIN SPECIFIC TRANSPOSON FAMILY.
AUTHOR(S): BINGHAM P M; KIDWELL M G; RUBIN G M
CORPORATE SOURCE: LAB. GENET., NATIONAL INST. ENVIRONMENTAL HEALTH SCI., RESEARCH TRIANGLE PARK, N.C. 27709.
SOURCE: Cell (Cambridge, Mass.), (1982) 29 (3), 995-1004.
CODEN: CELLB5. ISSN: 0092-8674.
FILE SEGMENT: BR; OLD
LANGUAGE: English

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